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PAROXYSMAL TACHYCARDIA, ACCOMPANIED BY THE VENTRICULAR FORM OF VENOUS PULSE.

By THOMAS LEWIS.

(*From University College Hospital Medical School.*)*

The ventricular form of jugular pulse may be defined as a type of venous pulsation, in which no constant and prominent waves present themselves, at such periods as represent the diastoles of the ventricle.

It has been regarded as significant of the absence of auricular contraction during the diastole of the heart; but it is now known that it may be found where the auricular systole, falling at its proper instant during the cycle, fails to affect the venous pressure appreciably or at all.[2] The events occurring in the auricle when the venous pulse is of the ventricular form cannot be gauged from this type of pulsation itself; for when it is present the mechanism of the auricle may be one of the following forms:—

1. Contraction, at the normal instant in the cycle, of an auricle which is in a state of distension, or the power of which is damaged in some other fashion.
2. Fibrillation of the auricle.
3. Simultaneous contraction of auricle and ventricle.

In a previous communication[2] the relationships of such abnormal auricular activities were discussed, and certain conclusions were formed in regard to them.

At that time it seemed clear that auricular fibrillation is never accompanied by regularity of the ventricle, except when it is complicated by auriculo-ventricular heart-block; and it appeared reasonable to assume that, where the ventricular form of venous pulse is present *and the pulse is regular* and of normal or increased rate, the auricle is not in a state of fibrillation.

The ventricular form of phebogram, associated with *regularity* of the pulse, may conveniently be grouped under three headings:—

(*a*) Where the pulse is slow and auricular fibrillation and heart-block are present.

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* An investigation carried out under the tenure of a Beit Memorial Research Fellowship.

(*b*) Where the auricle is distended and its contractions are ineffective in raising venous pressure ; the pulse being of normal or somewhat increased rate.

(*c*) Where the pulse is greatly increased.

It is under the last heading that a by no means uncommon type of paroxysmal tachycardia falls, of which a number of polygraphic curves have been obtained (Mackenzie,[5] Hewlett,[1] Lewis[2]). But the interpretation of the mechanism of the heart-beat in such cases has never been more than speculative, for hitherto no electrocardiograms have been won. It has been suggested that paroxysms of *regular* tachycardia, accompanied by the ventricular type of venous pulse, may arise, as a consequence of simultaneous auricular and ventricular systole, either in response to impulses originating in the junctional tissues,[6] or as a result of altered conduction, and the coincidence of an auricular systole with the ventricular systole of a preceding cycle[7] ; or, lastly, as the outcome of a ventricular tachycardia retrograde to the auricle.[2]

A case is now described of the type under discussion because it is hoped that it may throw light upon these questions, and in justification of the position adopted, that a rapid pulse which is *regular* is never present when the auricle is fibrillating, even though the ventricular form of venous pulse is found.

I am indebted to Dr. J. Rose Bradford for the opportunity of studying this patient.

The clinical history presents few features which have not been frequently duplicated by previously recorded cases of paroxysmal tachycardia ; therefore it is essential to report only the more salient symptoms and signs which were noted.

J. W. was admitted to University College Hospital on April 1st, 1910. A Hebrew of 28 years, and by trade a cook, he was carried to hospital in a critical state, and subsequently gave the following history. Rheumatic fever and chorea, it was stated, had never occurred either in himself or in his family ; apart from an attack of typhoid fever several years before admission, he had been healthy. Whilst mounting a flight of stairs he was seized with acute pain in the chest and distress of breathing. The pain was felt over the left half of the chest, the left abdomen, and eventually over the left arm and the left leg. He had never experienced a similar attack and its onset was not foreshadowed.

His condition when first examined gave rise to anxiety. The distress was evident and the collapse considerable. He complained of acute suffering, great pain in the precordial region, shooting into the neck and left arm ; a feeling of compression in the lower regions of the chest ; and a powerful throbbing in the neck. The skin was pale, cold and moist. He half reclined in bed ; restlessness was prominent and he moaned incessantly ; salivation was copious and he experienced nausea. The

respirations were rapid and the pulse was counted at between 200 to 210 beats to the minute. Cyanosis was not present. The heart sounds were tic-tac; no murmurs were audible; the dulness was increased to right and left; the impulse was diffuse in the fifth and sixth spaces in the nipple line. The chest was extremely sensitive to slight pressure and friction.

The attack of tachycardia which was present at his admission continued with its accompanying symptoms for eight hours, during which time the liver tended to enlarge, the heart dilated further, and venous engorgement became more evident. The attack ended abruptly and with immediate relief.

The subsequent history of the patient, during his long stay in the hospital, evolves itself into a series of attacks and the intervening intervals of rest. The paroxysms were frequent during the first two months of his stay and their duration varied from a few minutes to 48 hours. His freedom at a later date was attributable to rest, for the crises recurred when he first left bed. A number of the paroxysms originated in straining at stool. The onset was invariably sudden and the offset was always as abrupt. On several occasions the paroxysm ceased within a quarter of an hour of the injection of five drops of Cloetta's digalen or $\frac{1}{100}$ grain of strophanthine; on other and more numerous occasions the drugs were entirely without effect. Many means were resorted to, including ice packs and vagal pressure, but it was impossible to assert that a remedy had been found.

The condition between the paroxysms.

The patient was an extremely neurotic subject, poorly built and indifferently nourished; he presented no physical signs other than those discoverable in the heart. The lungs and abdomen were normal; there was never a sign of dropsy. The heart was enlarged. The right limit lay three-quarters of an inch and the left $4\frac{1}{2}$ inches from the middle line. The first sound was accentuated, the second normal. A distant presystolic murmur was present on many occasions, but varied in distinctness. A thrill was felt at the apex on one occasion only. The mechanism of the heart-beat as portrayed by polygraphic curves is shown in Fig. 1.

The pulse was regular as a rule, though at times when the usual rate of 70 to 80 was reduced to 50, sinus irregularity was noticed. The *a-c* interval was the full 0·2 sec..

The electrocardiograms are shown in Fig. 9, *I*, *II*, *III*. The lead from right arm and left leg (Fig. 9, *I*) is characteristic of mitral stenosis. P is of excessive height and bifurcates. The P-R interval is somewhat increased, measuring 0·18 to 0·20 sec.. In the lead from left arm and left leg (Fig. 9, *II*) R shows a notch on the upstroke. In the lead from right arm and left arm (Fig. 9, *III*) it shows a notch on the downstroke. The

conformation of these curves will be more specially referred to in the sequel. Occasional irregularities were felt at rare intervals, but none were recorded.

The condition during the attacks.

The symptomatology of the patient and many of the physical signs present during the paroxysms have been given. It remains to describe the mechanical and photographic records.

The polygraphic curves.—An example of polygraphic curves is shown in Fig. 2. The venous curve is of the ventricular form, the upstroke of

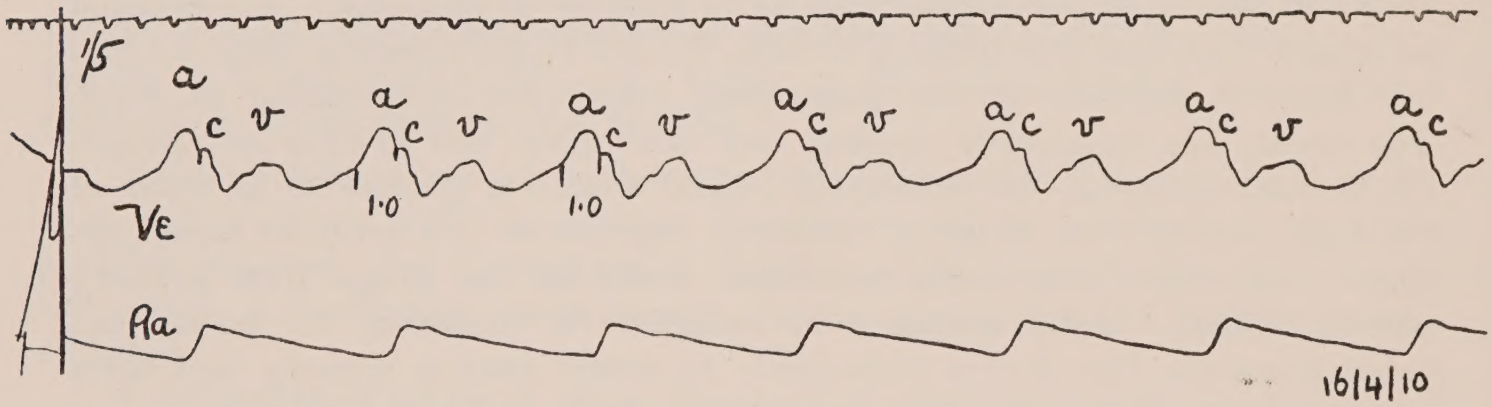

Fig. 1. Polygraphic curves taken during a period intervening between paroxysms, and showing the normal sequence of heart contraction, as indicated by the *a*, *c* and *v* waves in the jugular pulse. The *a*-*c* interval is 0·2 sec.. The time marker in this and succeeding curves is in 0·2 sec..

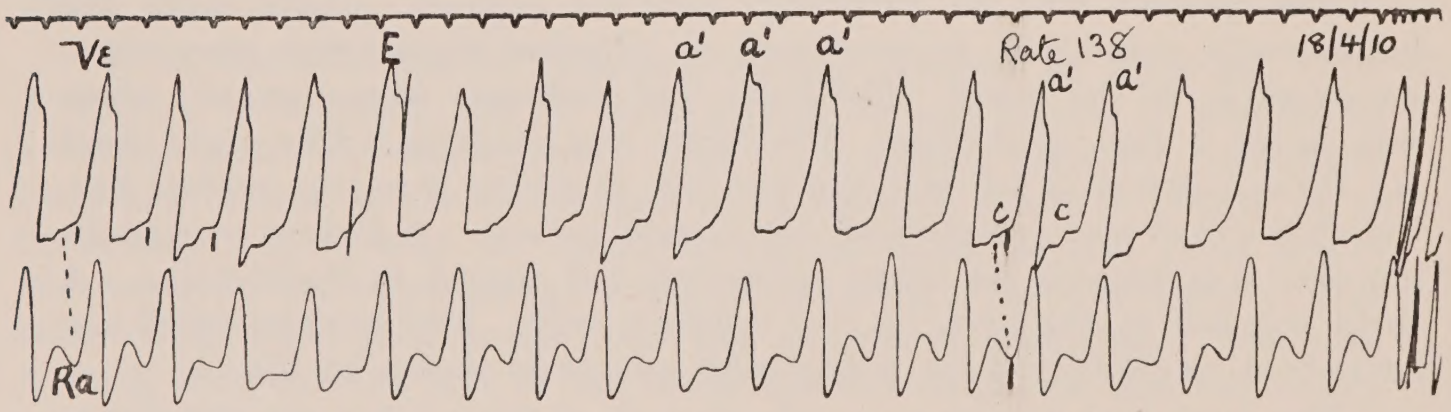

Fig. 2. Polygraphic curves taken during a paroxysm; the rate is 140; the venous pulse is of ventricular form, the large waves marked *a'* fall entirely within the limits of ventricular systole (E). The upstroke of *a'* occurs a little later than the point representing the onset of carotid pulsation.

the high peak coincides with the onset of the radial pulsation, or in other curves falls slightly before it; that is to say, the carotid upstroke falls slightly before or with the upstroke in the venous tracing. The complete wave has a duration of 0·2 sec., a little less than the duration of ventricular

systole as measured in the electrocardiographic curves. The beginning of the downstroke falls approximately at a point calculated as representing the bottom of the dip of the dicrotic in the carotid. The radial pulse is quite regular and markedly dicrotic.

The electrocardiograms.—A number of these curves were taken at different times and with different leads. They are shown in Fig. 9, *IV-VIII.* They are characterised by their regularity from beat to beat. There is no sign of irregular oscillation. Each peak and depression is duplicated from cycle to cycle.

Fig. 9, *I V* shows the curve obtained by leading from right arm and left leg. It consists of a tall peak R of similar duration to that of the similar lead during the slow periods (Fig. 9, *I*). It is succeeded by a deep depression, a further and gradual elevation and a last gradual depression. Then the next beat begins. The lead from left arm and left leg is shown in Fig. 9, *V*. It consists of a peak R, notched on the upstroke (marked ⊙). It is followed by a depression marked T. In no portion of the two preceding photographs is the curve flat; at no point can it be spoken of as isoelectric. The lead from right arm and left arm (Fig. 9, *VI*) presents a peak R with a notch upon the downstroke. It is succeeded by an upright after-swing T. The separate leads were adopted on each and all of the dates which the curves bear, but with the exception of the lead from right arm and left leg, an individual lead showed no conspicuous alteration. Two photographs of the lead from right arm and left leg are given because they are examples of paroxysmal curves at slightly different rates, namely 132 and 168. They differ in that with the faster rate the diastolic portion of the curve is curtailed and in that the latter is straighter with the accelerated heart-beat (Fig. 9, *VII*). A last lead is shown in Fig. 9, *VIII*, and it was taken from the junction of the sternum with the second costal cartilage and from the apex beat.

The interpretation of the curves obtained during the paroxysms.

Before proceeding to a description of the remainder of the tracings obtained from this patient, it will be profitable to discuss the significance of the paroxysmal curves themselves.

The venous curves are of the ventricular form, but two peculiarities call for remark. The excursion is of unusual amplitude and considered alone, the paroxysmal phebograms awaken a suspicion that the forcible venous pulsation, a pulsation readily appreciated with the finger, is not of ventricular origin, for curves of this amplitude and steepness and resulting from ventricular contraction are rarely if ever encountered except where there is great enlargement of the right heart and cavernous distension of the venous tributaries. The question as to whether auricle and ventricle

are contracting simultaneously suggests itself, for when a mechanism of this character is present similar venous tracings are met with.

The second peculiarity is one of less consequence. The upstroke in the venous curve does not coincide with the carotid but with the radial upstroke. This is of lesser significance, for the same time conditions are occasionally encountered, when it can be shown that the auricle is fibrillating.

The electrocardiograms.—Both the radial and electrocardiographic curves clearly show that the mechanism of the heart is regular during the paroxysms ; and it may be held, I think with justification, that where a ventricular rhythm is regular the impulses which give rise to it are generated in a single focus. But at the present time I would not use this proposition as an evidence of the nature of the mechanism, for it is more desirable that the case should be utilised in confirmation of the view that, where the pulse is regular and rapid, the auricle is not fibrillating. That such is actually the case is at once evident, for in none of the leads is there any trace of the fibrillatory oscillation. The absence of fibrillation and the similar mechanism from one cycle to the next is shown by the absolute repetition of the individual electric curves ; each beat is productive of a similar picture. The rhythm is consequently the result of impulse formation at a single focus, and it remains to locate the seat of this stimulus production. Taking the heart musculature as a whole, a large part of it may be eliminated at once by studying the electrocardiograms. The impulse formation is of supra-ventricular origin. The last conclusion is arrived at from the general conformation of the several curves in the slow and rapid rhythms and their comparison, and especially as a result of an examination of the curves given by separate leads. In the leads from right arm and left leg (Fig. 9, *I* and *IV*) the ventricular complexes differ during slow and fast rhythms respectively in that an inversion of T is present during the latter. A similar change has been reported in experimental auricular tachycardia, and the change is apparently associated with demonstrable conductivity changes in the heart.[3] The slight prolongation of P-R interval (Fig. 9, *I*) is in accord with these observations, and, as will be seen, depressed conductivity was present in other curves from the same patient. (Fig. 10, 11 and 12.) A similar inversion of T is observed in the leads from left arm and left leg (Fig. 9, *V*), but in this figure also the remainder of the curve is a remarkable duplicate of that of Fig. 9, *II* ; the notch on the upstroke of R (marked ⊙) is repeated. The resemblance in leads from right arm and left arm is striking (Fig. 9, *III* and *VI*). R and T are of the same type in both slow and paroxysmal curves, and a notch (marked ※) is present upon both. The general conformation of the curve in Fig. 9, *VI*, is in itself evidence of the supra-ventricular origin of the beat, which gives rise to such a curve ; and the special base apex lead from the chest wall (Fig. 9, *VIII*) provides additional support for the view. There is consequently no hesitation in pronouncing

the rhythm of the paroxysm as arising in a part of the musculature which lies above the division of the A-V bundle, and attention may be henceforth confined to the bundle itself and to the auricular tissue. Before pursuing the subject of localisation, the remainder of the curves will be described.

The curves obtained at the termination of the long paroxysms.

The four *polygraphic curves* (Fig. 3-6) were taken at one sitting and within a few minutes of each other; the end of a paroxysm of several hours' duration is shown in Fig. 3. It is succeeded by three brief runs of the quick beats, with a return to the normal sequence in each instance. A similar mechanism is shown in Fig. 4, which is a continuation of the last curve. At the end of the tracing a paroxysm, approximately 10 minutes in length, has its onset. At its termination the two remaining curves were secured. Fig. 5 and 6 are examples of the slow normal rhythm, interrupted by premature beats.

In the investigation of all cases of paroxysmal tachycardia the end curves are of the utmost value and throw considerable light upon the nature of the disorder with which the heart is affected. As in previously reported cases, so in this patient, the paroxysms end in post-paroxysmal pauses, the duration of which is an indication of the ectopic character of the new rhythm. The normal stimulus production has been disturbed by the interference of pathological impulse formation at a point distant from the pacemaker. The end pauses of the long paroxysm shown in Fig. 3, and of the short paroxysms succeeding it, are of duration $\frac{5 \cdot 7}{5}$ to $\frac{5 \cdot 8}{5}$ sec. in the radial curve (the auricular pauses are $\frac{3 \cdot 4}{5}$, $\frac{3 \cdot 9}{5}$, $\frac{4 \cdot 0}{5}$, $\frac{4 \cdot 0}{5}$ sec.). The majority of the ventricular pauses following single premature beats (Fig. 5 and 6) are of duration $\frac{5 \cdot 5}{5}$ to $\frac{5 \cdot 9}{5}$ sec. (the corresponding auricular pauses $\frac{3 \cdot 9}{5}$, $\frac{4 \cdot 0}{5}$, $\frac{3 \cdot 6}{5}$, $\frac{4 \cdot 4}{5}$ sec.). The mechanism though lacking absolute regularity is similar in one case and the other. Following the paroxysms and following the single premature beats, the length of the pause in the auricular curves is greater than the distance between adjacent beats of the normal rhythm in Fig. 5 and 6 (the latter varies from $\frac{3 \cdot 0}{5}$ to $\frac{3 \cdot 5}{5}$ sec.).

The venous curve shown in Fig. 3 is of particular value, for it allows a comparison of the amplitude of the paroxysmal curves and the *a* and *c* waves accompanying beats in which the contraction is sequential. The second ventricular beat which follows the post-paroxysmal pause probably belongs to the normal rhythm; it is followed by four premature beats, each accompanied by a tall venous wave marked *a'*. Now it is evident that these waves are not of purely ventricular origin for the remaining *c* waves of the curve are extremely insignificant. It is rational to assign to them a new factor of production, and the increased amplitude can be explained by supposing that a premature auricular contraction has occurred.

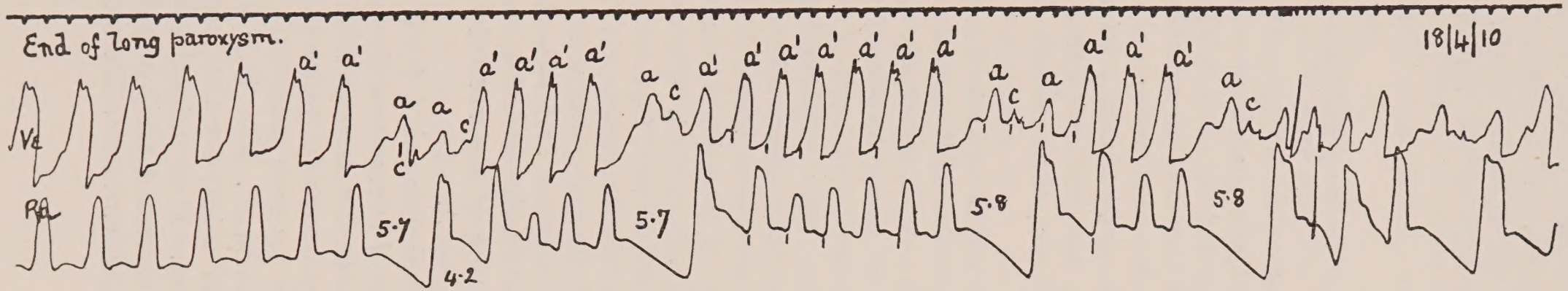

Fig. 3. Polygraphic curves showing the termination of a long paroxysm of the same nature as that shown in Fig. 2. The end of the paroxysm is marked by a pause at the return to the normal sequence, and is followed by the occurrence of three short paroxysms of a similar character.

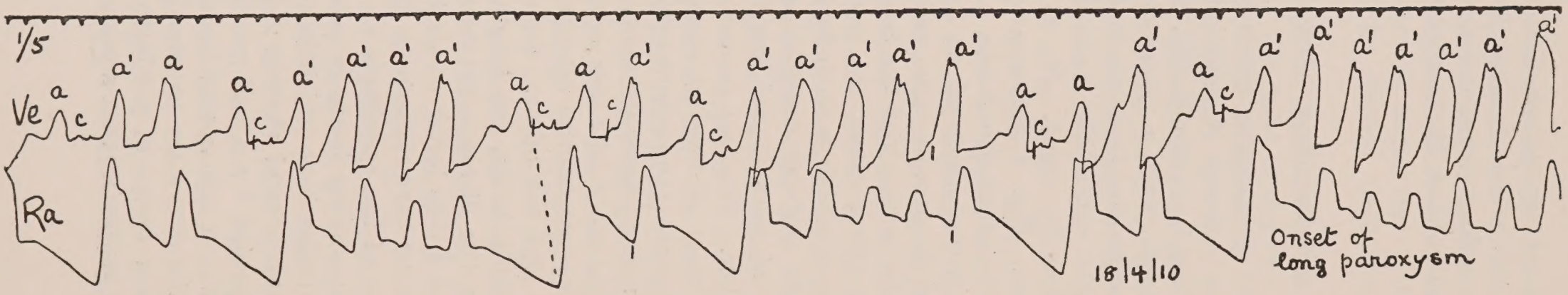

Fig. 4 is a continuation of the preceding figure, and shows the irregularity which follows the end of a paroxysm, as a result of interruption of the normal sequence by beats of similar form to those of the paroxysms themselves. At the end of the curve a fresh and prolonged paroxysm commences.

And this explanation is fully justified by a detailed examination of the curve. The height of the new wave exceeds that of the normal *a* waves, but each is accompanied by a ventricular systole. The new waves vary also in their relationships to the radial beats, in itself a conclusive argument against their ventricular origin; further, the closer the coincidence with the ventricular contraction the higher is the resultant wave. The conclusion that the new waves are of auricular origin is beyond question.

The interpretation of this curve and the continuation of it (Fig. 4) is fully borne out by the succeeding tracings, in which single interruptions of the slow rhythm are seen. Examine Fig. 5 and 6. It may be said that high waves also occur as accompaniments of beats following long pauses. But there is good reason for this variation: it is due in these instances, as in those previously discussed, to synchronous auricular and ventricular contraction; in the last instances the ventricle escapes and beats are present which belong to an ideo-ventricular rhythm. Where a comparison with electric curves provides an easy solution of the mechanism (Fig. 10, 11 and 12), positive evidence of simultaneous contraction is forthcoming.

The electric curves (Fig. 10, 11 and 12) are examples of a large number of photographs, and are selected to illustrate special points connected with the interpretation of the mechanism as presented by Fig. 5 and 6. The venous and electric curves referred to were taken within half an hour of each other. In the radial curves of Fig. 5 and 6 the beats occur in groups of two and three. Similar groups are shown in the electric curves. In Fig. 10 two complete groups of three ventricular cycles are shown. Each heart cycle is accompanied by P, R and T variations. In each group the P-R interval increases from 0·6—1·7 sec., and the last beat, which is of anomalous form, is succeeded by a long pause. The relation of the P and R variations is similar to that of the *a* and *c* waves in Fig. 6. The shortening of the P-R interval after a pause, to $\frac{0\cdot8}{5}$ sec., is conspicuous. In other curves it is still greater, and the interval diminishes until P and R partially coincide (Fig. 11), or until the auricular complex is completely buried in the ventricular complex (Fig. 12). As all stages of shortening are present, it is impossible in certain given instances to ascertain whether the ventricular beat is a response to the auricular, or whether it is generated in the ventricle. It is probably ideo-ventricular in Fig. 11. It is certainly ideo-ventricular in Fig. 12; the last beat in the curve consists of a superimposition of auricular and ventricular complexes. Thus the coincidence of As and Vs, suspected after an examination of the venous curves, is demonstrable.

The paroxysmal cycles are known, from the consideration of venous and electric tracings, to consist of simultaneous auricular and ventricular systoles; but this is not obvious in the electric curves alone (Fig. 9). Neither can the auricular contractions, which are recognised as falling with the

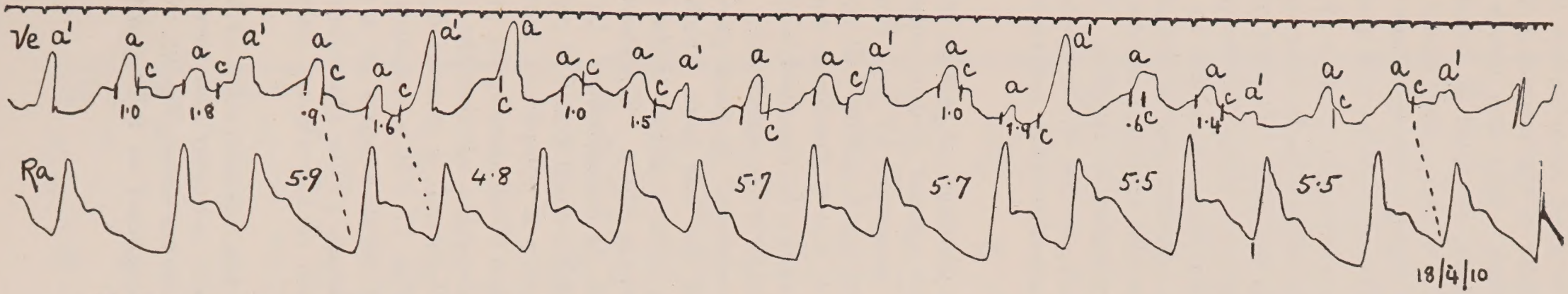

Fig. 5. A curve taken at the cessation of a paroxysm shown at the end of Fig. 4. The degree of irregularity is relatively small. There are interruptions of the normal sequence by beats of paroxysmal type. The examination of the *a-c* intervals shows conductivity changes, which are more fully described in the text. At one point in the curve an escaped ventricular contraction is seen.

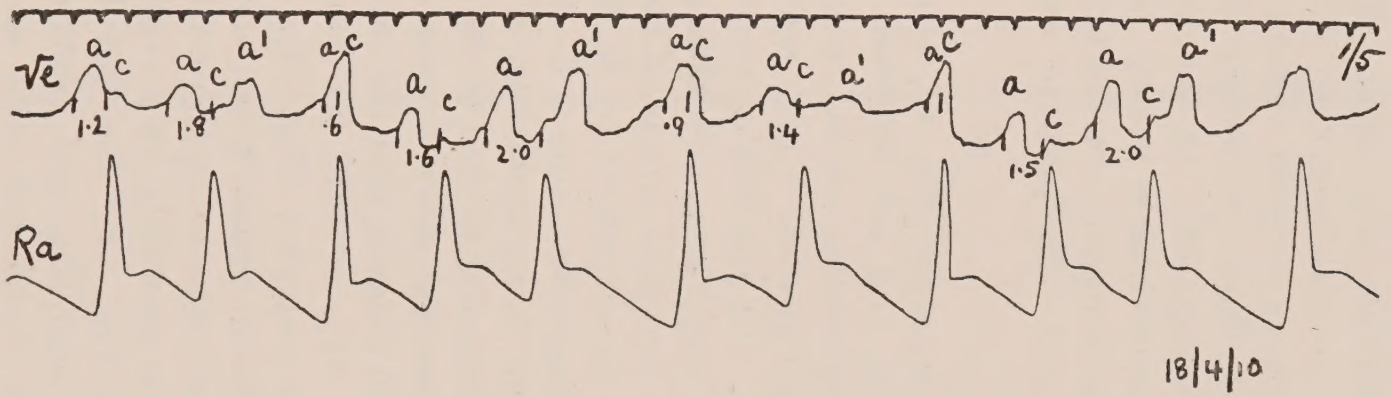

Fig. 6. A curve of the same sort, in which single premature contractions interrupt the auricular sequence at less frequent intervals. Evidences of the escape of the ventricle and of conductivity changes are clear in the venous curve.

last beats of the groups (Fig. 5 and 6 and Fig. 10, 11 and 12), be identified in the electric curves. The reason of the failure of the premature P variations (single or paroxysmal) in the electric curves is clear. The auricular contractions take an abnormal course in the auricle; that is to say, they commence at a point in the musculature other than the pace-maker. Under these circumstances the electric changes which accompany such contractions are anomalous, and in the present instance the form of the curve to which they give rise is unknown. That auricular contractions originating in an abnormal fashion give rise to abnormal electric variations is known from a study of the experimental facts in regard to them.[3] In this case the abnormal Ps are probably of a type approaching the iso-electric position. It was hoped that a clearer appreciation of the form of anomalous Ps would be obtained by comparing the cycles in various leads, and with slightly divergent heart rates (Fig. 9, *IV* and *VII*), but the analysis has been found impracticable. We remain satisfied that the contractions of auricle and ventricle are simultaneous, although the identification of the former is impossible in the electric curves.*

Returning to the mechanism of the paroxysms, and knowing that each cycle consists of a synchronous As and Vs, there are two alternative interpretations of the events open to us. It may be supposed that the auricle and ventricle contract together in response to a single stimulus originating between them, or that each ventricular contraction is a response to the auricular systole which coincides with the preceding ventricular contraction. Either interpretation meets the facts of the case in so far as we have yet considered them. The instants at which As and Vs fall in the venous curves are fully determinable, and we have to consider the functional relationships of the separate beats.

For purposes of illustration I have diagrammatised the two interpretations of the first short paroxysms of Fig. 3 (in Fig. 7, *1* and *2*) and the first six ventricular cycles of Fig. 6 (in Fig. 7, *3* and *4*).

(1) Dealing with the first interpretation, namely simultaneous As and Vs, as a result of As falling back upon Vs (Fig. 7, *1* and *3*), it will be obvious that if this explanation is adopted, it necessitates the assumption that all single premature auricular contractions are blocked. But it likewise involves the unreasonable assumption that the last beat of each paroxysm, be it short or long, is blocked too.

(2) Adopting the second interpretation (Fig. 7, *2* and *4*), namely simultaneous contraction of A and V as a result of a single impulse formed between them, we have to allow that the auricular contraction of the normal sequence directly preceding the onset of the paroxysm is blocked, or if this is not blocked, then the first impulse starting the paroxysmal contraction gives a response in the auricle alone.

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* An instance in which venous curves show what electric curves will not demonstrate.

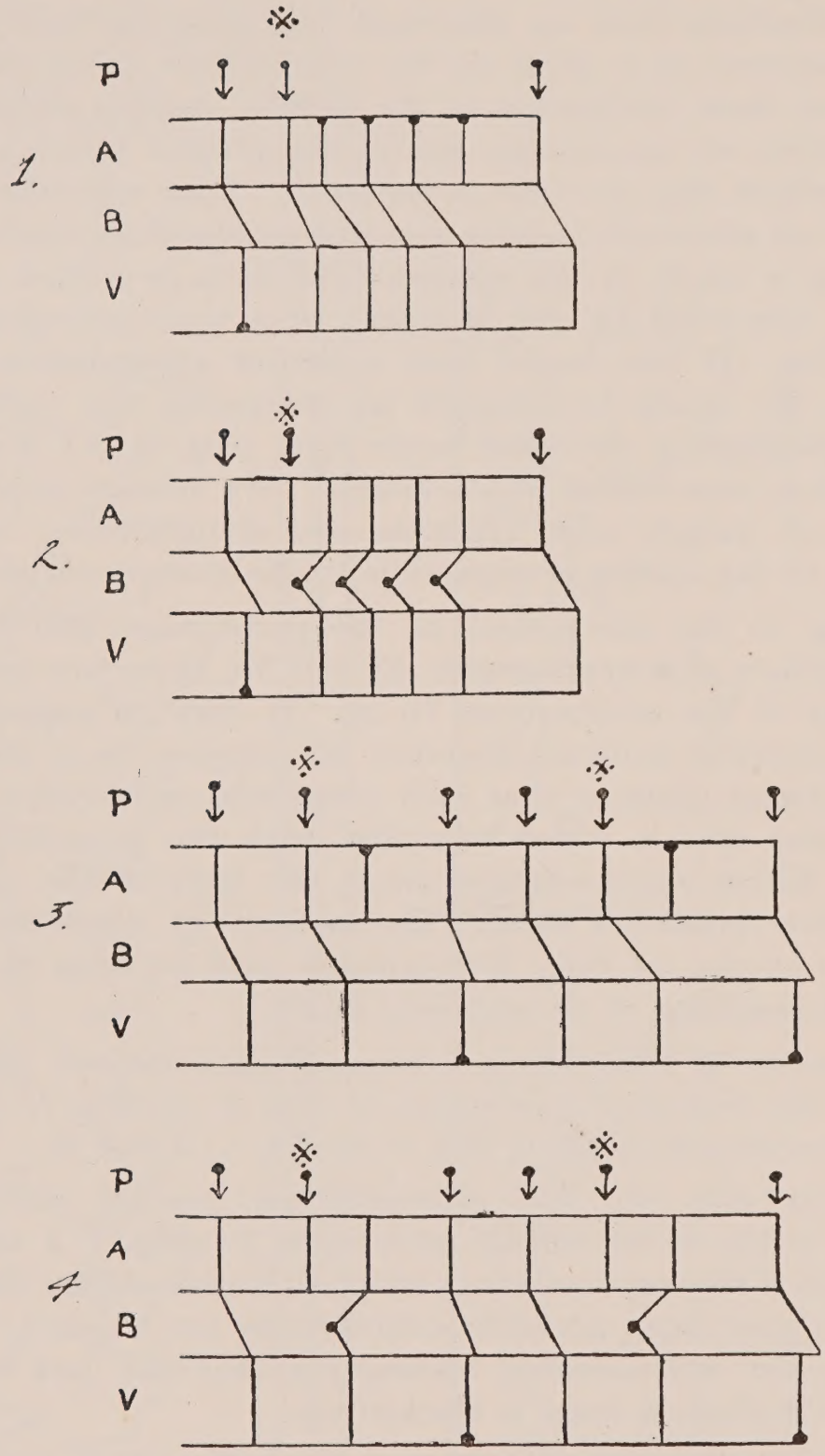

Fig. 7. Four diagrams illustrating the possible interpretations of mechanism in two portions of the preceding curves. Fig. 7, *1* and *2* are alternative explanations of the first short paroxysm of Fig. 3. Fig. 7, *3* and *4* are alternative and parallel explanations of the first five beats of Fig. 6. The arrows drawn above each diagram represent impulses which descend from the pacemaker and give rise to auricular contractions. A represents the auricle, B represents the junctional tissues, and V represents ventricle. The black dots are intended to represent the point at which the impulses may be supposed to arise.

The facts will not permit a positive decision in one way or the other, but on the whole they are in favour of the second explanation. No appreciable change has been found in the relationship of As to Vs during the paroxysms with considerable divergence of rate, and no ventricular beat has ever been dropped at such times. Finally, we can only conclude that we are dealing with a supraventricular form of paroxysmal tachycardia, in which the contraction of auricle and ventricle is simultaneous, and that of two propositions (delay in conduction from A to V, and impulse formation between A and V), which might be adopted in explanation of the phenomena, the second appears to be the more reasonable.

Certain features of the curves remain for brief discussion or mention.

(1) The last ventricular complex of each separate group in Fig. 10, 11 and 12 is of anomalous form. T is partially inverted. Such beats are of a form transitional between the normal complexes on the one hand and the paroxysmal complexes on the other. The alterations in a ventricular complex resulting from a premature auricular contraction have been described in detail in an article to the last number of this *Journal*,[3] but evidence was produced which showed that the prematurity of the auricular contraction is not the sole cause of the alteration in the ventricular curve. It appears to associate itself with two phenomena; the first of which is the close proximity of the premature beat and that which precedes it (brought about in the present instance by variation in the lengths of the As-Vs intervals), and the second of which is the presence of demonstrable changes of conductivity in some portion of the heart muscle. Fig. 12, taken at a fast rate, is given that the comparison between the single anomalous complex and the beats of the paroxysmal type may be more readily compared. There is no reason to suppose from the general conformity of the single anomalous beats that they have arisen other than in response to the preceding auricular contraction.

(2) The single atypical beats of Fig. 10, 11 and 12 are of interest in showing a gradual decline of T. It is some while before the curve becomes horizontal or isoelectric. The usual experience in dealing with premature complexes is to find the total length of an individual ventricular complex equal, within small errors of measurement, to the complex of the normal rhythm.[4] In this instance there is a marked divergence from the customary findings, but the cause of the extension of T is quite obscure. It cannot be assigned to activity of the auricle; the prolongation is too great. A similar condition is found in the paroxysms themselves. In the leads from right arm and left leg (Fig. 9, *IV* and *VII*), no portion of the curve is isoelectric for any length of time. Here, again, the meaning of the phenomenon is not ascertainable.

The ventricular form of venous pulse in association with retrograde heart-beats.

In the introductory paragraphs of the present communication it was stated that the ventricular form of venous pulse is to be anticipated when a paroxysm of tachycardia, originating in the ventricle, dominates the rhythm of the whole heart. I have met with two clinical instances in which ventricular paroxysms were present, but they were of brief duration and the venous curves were not obtained.

A polygraphic curve taken from an anæsthetised dog, and showing a complete paroxysm of ventricular tachycardia is shown in Fig. 8. The paroxysm was induced by interrupted stimulation of the ventricle; the chest wall was closed at the time. The normal sequence of events is shown in the opening cycles of the curve. The first interruption consists of a single premature ventricular beat, which fails to affect the femoral curve. One cycle of normal sequence follows, and a ventricular paroxysm of 15 beats succeeds it. The first four auricular peaks, which occur after the onset of the paroxysm, are placed at regular intervals, and at the expected points in continuation of the preceding auricular rhythm. They vary in height according to the stage of ventricular systole at which they fall, being taller when coinciding with the earlier phases of ventricular contraction. The venous curve then assumes a perfectly regular appearance. The auricle and ventricle are now contracting together and at constant time relationships to each other. In other words, the ventricular rhythm has become regularly retrograde. The representative of auricular contraction is seen rising from the plateau in each cycle of the venous curve. That this peak is in reality the result of auricular contraction is known by comparing the two cycles marked x, x. In the venous curve they show a close resemblance to each other, differing only in the very slightly earlier appearance of *a* in the first of the two cycles considered. Its appearance in the first cycle marked x is not due to retrogression, but it is due to the expected response of the auricle to sinus impulse formation. On the other hand, it appears prematurely in the second cycle, and represents the first retrograde auricular contraction. The venous curve during the reversed mechanism, which is maintained from this point onwards, is of the ventricular form.

Summary.

A case of paroxysmal tachycardia is described in which the ventricular form of venous pulse was present, as a result of simultaneous contraction of auricle and ventricle.

The ventricular form of venous pulse is also found (experimentally) when reversed heart rhythm is present.

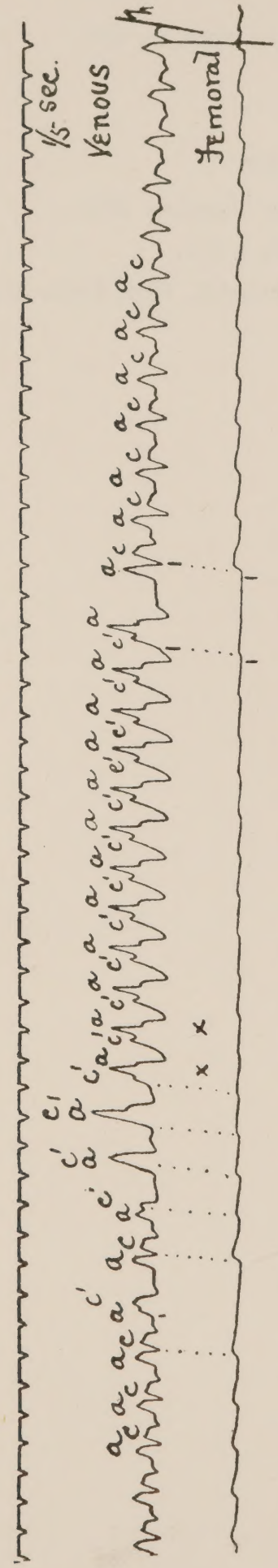

Fig. 8. A polygraphic curve from a dog, showing the form of the venous pulse when the rhythm of the heart is reversed and auricle is responding to ventricle.

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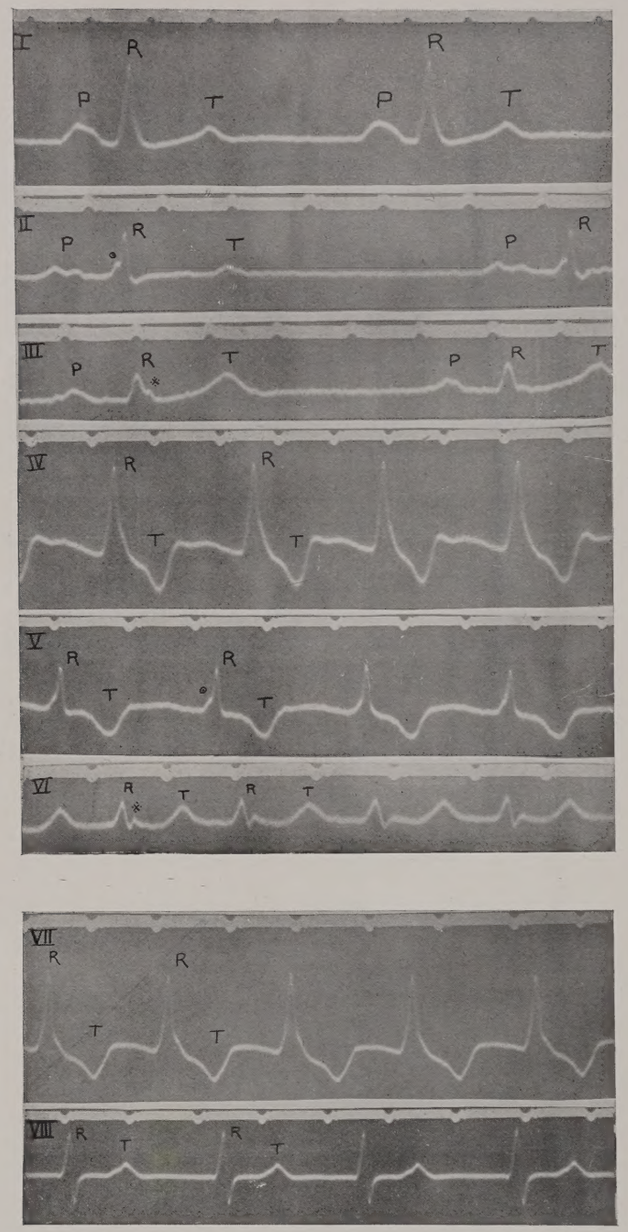

Fig. 9.

Fig. 9. × ½ linear.

I, 15-4-10. An electrocardiogram taken during the interval between two paroxysms. The P-R interval is 0·16 sec.. P is exaggerated, and in its outlines resembles auricular complexes obtained in other cases of mitral stenosis. The lead is from the right arm and left leg.

II, 3-5-10. The same as *I*; the lead is from the left arm and the left leg.

III, 3-5-10. The same as *I* and *II*; lead from right arm and left arm.

IV and *V*, 20-4-10, and *VI*, 24-4-10. Three curves taken during paroxysms with same leads as in *I*, *II*, and *III* respectively.

VII, 24-4-10. Lead from the right arm and left leg taken during a paroxysm and for comparison with Fig. 9, *IV*, in which the heart-beat is at a somewhat slower rate.

VIII, 20-4-10. Taken during a paroxysm, the lead being from the sternum at the junction of the second rib and from the apex.

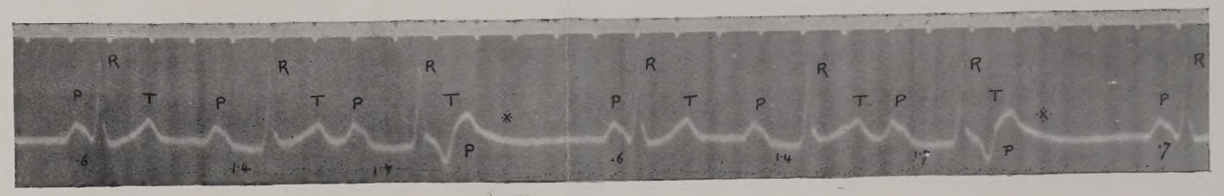

Fig. 10.

Fig. 10. 18-4-10. × ½ linear. Electrocardiogram from right arm and left leg taken shortly after the cessation of the long paroxysms, shown in the text, Fig. 3 and 4. The figure clearly shows the correctness of the interpretation of Fig. 5 and 6; it shows two beats of anomalous form at the termination of two groups of three beats. The P-R interval shows a notable increase during the progress of each separate group of beats.

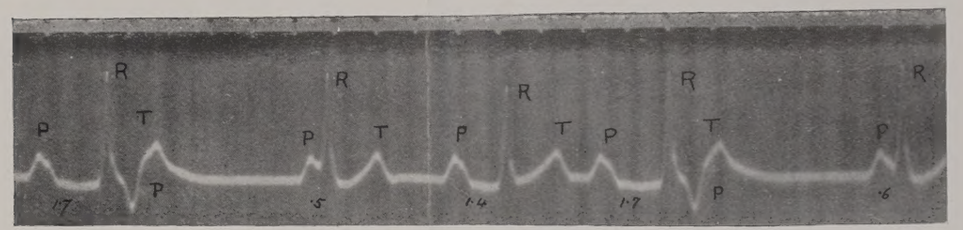

Fig. 11.

Fig. 11. 18-4-10. × ½ linear. A similar curve from right arm and left leg taken at the same time and showing a remarkable shortening of the P-R interval at the commencement of each group of beats. The interval is too short to permit the supposition of ventricular response to an auricular impulse.

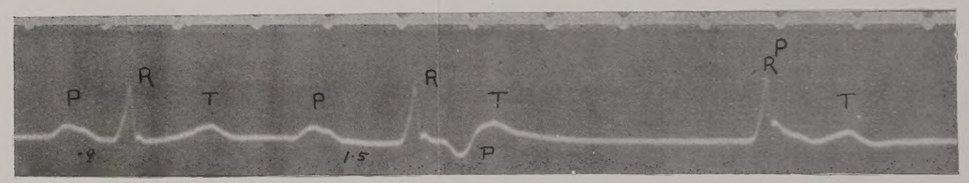

Fig. 12.

Fig. 12. 18-4-10. × ½ linear. A similar curve taken from right arm and left leg and at a higher speed for the purpose of comparison with the preceding curves. The third beat in the strip clearly demonstrates the escape of the ventricle and simultaneous contraction of auricle and ventricle.

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